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Atherosclerosis of the Aorta: A Risk Factor, Risk Marker, or an Innocent Bystander?

In a recent issue of the *Journal*, Meissner et al. (1) should be congratulated for carrying out a community-based transesophageal echocardiography (TEE) study of thoracic aortic plaque. However, because they included patients with descending aortic plaque (which is more prevalent than arch plaque), their negative conclusions regarding stroke are flawed. It is simply not plausible to consider plaque limited to the descending aorta as a cause of cerebral embolization.

Earlier studies of aortic *arch* plaque have found a 12% (2), 12% (3), and 13.7% (4) risk of cerebral embolization. This is 50% higher than the 8% risk of stroke in the "high risk" Stroke Prevention in Atrial Fibrillation (SPAF)-III study patients (5). In fact, if these "high risk" Stroke Prevention in Atrial Fibrillation (SPAF) study patients had no significant plaque in the aorta on TEE, their risk fell to 1.2%.

Although the investigators recognize this problem in their limitations section, we would like to re-emphasize that aortic *arch* plaque is associated with a high risk of stroke.

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doi:10.1016/j.jacc.2005.03.004

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REPLY

We appreciate the thoughtful comments of Drs. Tunick and Kronzon to our study (1), "Atherosclerosis of the Aorta: Risk Factor, Risk Marker, or Innocent Bystander?"

Plaques in the proximal descending aorta may theoretically embolize retrogradely, as previously suggested (2).

Although the correlation of plaques in the descending aorta with embolic cerebral events seems counterintuitive, location of aortic atherosclerosis has not been shown to correlate clearly with site of embolism. In the Stroke Prevention in Atrial Fibrillation (SPAF)-III study, precisely the same definitions were used as in our study. Almost all plaques were in the descending aorta, whereas all embolic events were cerebral, suggesting that plaques in the descending aorta do in some way correlate with stroke, as high-risk markers, or, invoking the possibility of retrograde embolism to some extent, as a direct source of embolism (3). Nonetheless, we did secondary analyses of stroke end points in subjects with only ascending and arch plaques (Table 5 in the Meissner et al. [1] study) and similar results were obtained as was commented upon in the discussion section. In our study, after adjustment for age and gender, the hazard due to plaque was no different from the baseline hazard.

Moreover, the results for a cerebrovascular event, in particular, were essentially the same whether or not the descending aorta was included or excluded in the analysis (hazard nonsignificantly increased about 80%, after adjusting for age and gender, for both models; Table 5 in the Meissner et al. [1] study). Further, before adjusting for age and gender, the estimates of the increase in the hazard of a cerebrovascular event were nearly the same both with and without the descending aortic plaques included.

Finally, the studies referenced by Dr. Tunick and colleagues were limited to patients with stroke who survived long enough to be enrolled (4), to those referred for transesophageal echocardiography (TEE) because of a specific indication (5), and to patients with atrial fibrillation (3), whereas ours were randomly selected community subjects without any indication for TEE. Our population would be more representative of patients seen in the community; consequently, they may not show the strong causal association between aortic arch plaque as reported in studies of highly selected (high-risk) patients.

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doi:10.1016/j.jacc.2005.03.005

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